Graefe's Arch Clin Exp Ophthalmol (1988) 226:206–212

Graefe's Archive
for Clinical and Experimental
Ophthalmology

Anterior ischemic optic neuropathy: classification of field defects by Octopus™ automated static perimetry

Oli I. Traustason [1], Steven E. Feldon [1,2], Jay E. Leemaster [1], and John M. Weiner [3]
Departments of [1] Ophthalmology, [2] Neurological Surgery, and [3] Medicine, University of Southern California School of Medicine, and the Estelle Doheny Eye Foundation, Los Angeles, California, USA

Abstract. Visual fields of patients with anterior ischemic optic neuropathy (AION) were classified according to quantitative criteria, using the Octopus™ perimeter. Although a significant altitudinal pattern of field loss was found in 55% of perimetric examinations, the "spared" hemifields routinely showed some loss of sensitivity. This finding, along with the diffuse loss of sensitivity in a high percentage of visual fields, indicates more extensive involvement of the circulation of the anterior optic nerve head than has previously been suggested. Furthermore, patients with diabetes mellitus alone were found to have a statistically separable pattern of visual field loss. The pathophysiologic implications of the visual fields in AION and their relationship to the clinical findings were investigated.

Introduction

Nonarteritic ischemic infarction of the anterior portion of the optic nerve (AION) is a well-known clinical entity. Clinical studies have identified typical patterns of field loss and animal models have been developed [2, 12], but the mechanism of ischemic optic neuropathy is still controversial.

Histopathologic correlations with clinical cases of AION suggest that the major damage to the optic nerve occurs in regions adjacent to the lamina scleralis [4, 12, 14, 16, 17]. This region of the optic nerve is supplied by the retinal, pial, and posterior ciliary circulation. However, the posterior ciliary arteries are generally considered to provide the critical circulation [1, 11, 18]. Hayreh [12] reported localized sectoral prelaminar infarction of the optic nerve with experimental occlusion of a single posterior ciliary artery; horizontal, vertical and temporal distributions of infarction were found. Anderson and Davis [2], on the other hand, found that ischemic optic neuropathy simulating the clinical disease required occlusion of multiple posterior ciliary arteries.

A clinical study to analyze patterns of visual field loss quantitatively in patients with ischemic optic neuropathy may determine the conditions that result in focal or diffuse optic nerve damage. The results of such a study are presented and etiological implications are discussed.

Offprint requests to: S.E. Feldon, Estelle Doheny Eye Foundation Building, 1355 San Pablo Street, Los Angeles, CA 90033, USA

Materials and methods

Patients

Forty-seven eyes of 37 patients studied at Estelle Doheny Eye Medical Clinic were diagnosed as having anterior ischemic optic neuropathy. The clinical criteria used for diagnosis included sudden onset of symptoms, stable visual field loss, and acute unilateral swelling of the optic nerve, which was replaced by pallor upon follow-up examination. Cases for which history of onset was uncertain or possibly progressive were also evaluated by computed tomography of the anterior visual system. Patients with constitutional symptoms such as weight loss, fever of unknown origin, polymyalgia rheumatism, scalp tenderness, or jaw claudication were excluded from the study. Sedimentation rate was obtained on all patients to screen for possible temporal arteritis. When clinical evaluation failed to differentiate idiopathic from arteritic anterior ischemic optic neuropathy, a temporal artery biopsy was performed.

Equipment

The Octopus™ automated perimeter consisted of a hemispheric screen of 0.5 m radius upon which a stationary Goldmann III target was projected in a randomized sequence. The threshold level of stimulus intensity at which the patient identified the target was recorded at fixed intervals across a designated region of the visual field. Reliability was determined as a percentage of false-positive and false-negative responses. Sensitivity was estimated by the variability on retesting of designated points (Program 31 only). Fixation was monitored by closed-circuit infrared-sensitive television. Responses to stimuli that occurred during blinks or altered fixation were suppressed. Information was stored on floppy disks. Hard copy was available both in numeric and symbolic (grey-scale) form. Details of the hardware and software strategy have been published elsewhere [26].

Procedure

Before each perimetric examination, the illumination of the background was standardized. The 76 test points (Program 31 or 33) within the central 30° field were spaced at 6° intervals and intersected both the horizontal and vertical meridians; appropriate near correction was used. Using the Delta program supplied with the Octopus, fields were analyzed statistically by quadrants, deleting the physiologic

Table 1. Summary of clinical data

Case/age/sex/eye	Visual acuity	Afferent pupillary defect	Color		Diabetes mellitus	Hypertension	Cataract extraction	Lower field minus upper field loss (dB)	*t*-value central 30°	Total field loss (dB)
			OD	OS						
1 /78/F /OS	20/100	+1	15/15	6/15	+	+	+	−16.7	−15.37	10.8
2 /58/M/OD	20/ 60	+2	10/15	10/15		+	+	−13.9	−12.53	11.2
3a/60/M/OD	20/ 70		10/15	7/15				3.7	1.31	14.6
3b /OS	20/ 30							−15.4	−8.96	7.7
4a/76/F /OD	20/100		0/15	6/15				10.3	5.33	5.2
4b /OS	20/ 50							−8.6	−8.67	6.5
5 /58/F /OD		+2	0/15	10/15				−2.4	−4.91	22.7
6 /53/F /OD	5/200	+2	8/15	15/15				−5.4	−4.39	11.7
7 /60/M/OD	20/200		9/15	11/15		+		−4.6	−4.09	9.9
8 /52/F /OD	20/ 25	+3	11/15	11/15		+		−5.2	−3.26	8.5
9 /43/F /OS	20/ 40	+1	15/15	0/15	+			−2.1	−2.84	25.7
10a/55/M/OD	20/200		0/15	0/15				0.2	−1.57	24.2
10b /OS	20/400							3.7	1.73	23.6
11 /65/F /OD	20/ 80	+1	0/15	15/15			±	0	−1.24	10.1
12 /67/M/OS	20/ 50	+2	10/15	3/15				−2.9	−0.66	2.2
13 /43/M/OD	20/ 30	+1	0/15	9/15	+	+		0.9	−0.38	8.4
14a/84/F /OD	10/200	+2	0/15	7/15		+		1.0	−0.13	16.8
14b /OS	20/ 80							1.7	0.47	16.4
15 /55/F /OS	HM	+3	11/15	0/15	+			1.2	0	25.7
16 /61/F /OD	20/400	+2	0/15	11/15			+	0.1	0.13	12.0
17 /50/F /OS	20/ 15		14/15	13/15				2.0	1.06	1.4
18a/67/M/OD	20/ 60		8/15	8/15	+			4.4	2.75	21.3
18b /OS	20/ 30							0.9	−1.03	23.6
19 /67/M/OS	20/100	+2	10/15	0/15		+		5.1	1.94	14.7
20 /47/F /OS	20/ 50		15/15	10/15				4.3	2.01	2.6
21a/70/M/OD	4/200	+3	0/15	6/15		+		7.5	4.46	19.9
21b /OS	20/ 40							11.3	2.14	6.7
22 /83/F /OS	20/200	+2	9/15	0/15	+	+		4.1	2.36	20.0
23a/74/F /OD	20/ 70		4/15	1/15	+			8.8	4.04	10.2
23b /OS	20/100							6.6	3.06	11.5
24 /46/F /OS	20/ 60	+3	14/14	9/15				5.6	3.71	7.8
25 /57/F /OS	20/200	+1	10/15	0/15			+	8.6	4.35	17.8
26a/55/F /OD	20/ 50		1/15	7/15	+	+		19.3	13.49	14.1
26b /OS	20/ 80							−2.1	6.09	9.9
27 /52/F /OS	20/ 30	+2	12/15	11/15				20.4	12.93	11.6
28a/66/M/OD	20/ 40		9/15	0/15				21.1	25.51	13.9
28b /OS	6/200	+2						20.0	16.77	13.9
29 /53/F /OS	5/200	+1	13/15	1/15				24.7	38.36	13.7
30 /84/M/OS	CF	+4	9/15	0/15		+				
31a/49/M/OD	20/ 60	+1	10/15	12/15		+				
31b /OS	20/ 20									
32 /58/M/OS	20/200	+1	14/15	12/15						
33 /73/F /OD	1/200	+2	0/15	8/15	+		+			
34 /75/F /OS	HM		13/15	0/15						
35 /57/F /OS	20/ 50	+2	12/15	4/15		+				
36 /64/M/OS	HM	+3	12/15	0/15						
37 /74/M/OS	3/200	+1	12/15	0/15						

blind spot. Analysis was also performed as a function of eccentricity from fixation, divided into 10° intervals from 0–60°. The arcuate region was defined as the region between 10° and 20° from fixation. Average sensitivities (dB) for individual full fields and field segments of patients were compared with those from age-matched controls, defined in the Octopus Visual Field Atlas [22].

Results

Analysis of clinical data

A summary of clinical data is shown in Table 1.

Age and sex distribution

The age range of patients studied was from 43 to 84 years, with a median of 60 years and a mean of 61.9 years. There was no difference in average age between females and males. Twenty-two of 37 patients were female, a 1.3:1 ratio of females to males.

Systemic illness

Controlled diabetes mellitus was present in 9 of the 37 patients, one of whom had juvenile-onset diabetes, and one of whom had steroid-induced diabetes; the remainder were

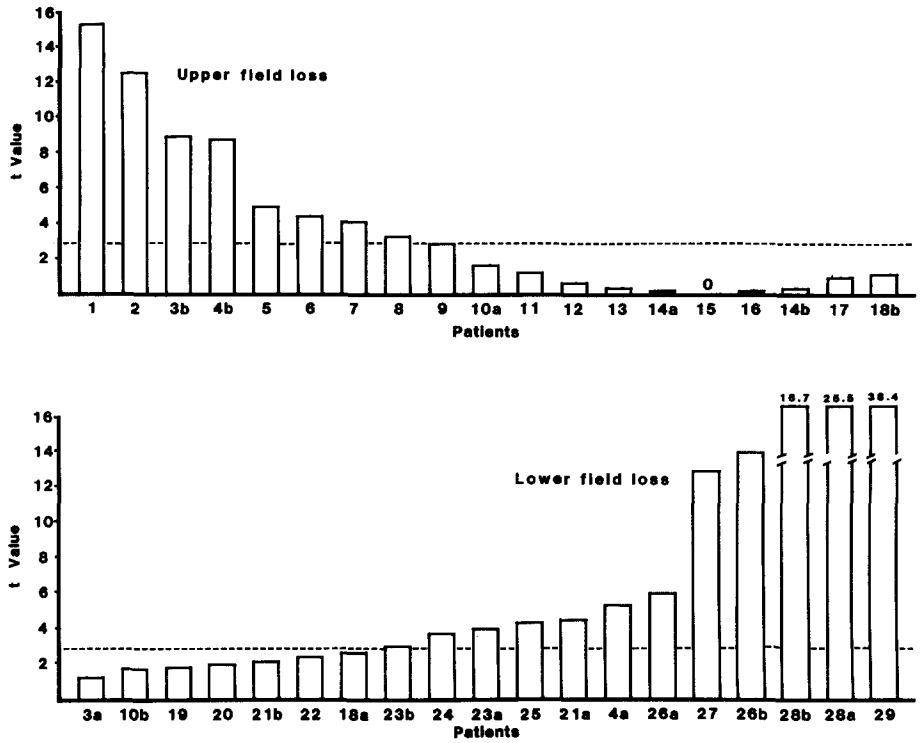

Fig. 1. Severity of altitudinal field loss was determined by comparing upper and lower fields using Student's *t*-test. Significant altitudinal character ($t > 2.9$; < 0.05) is shown by the *dotted line*. Fields are arranged from most significant upper field loss to most significant lower field loss, as determined by value of *t*. Patient numbers refer to Table 1

adult onset, controlled by either diet or oral medication. The juvenile-onset diabetic was the youngest patient in this series, being 43 years of age. The average age of the diabetics (63.4 years) was, however, not significantly different from that of the total population. All patients were evaluated with random blood sugars as a screen for diabetes.

Thirteen patients had a history of hypertension. A blood pressure reading was obtained on all patients. No one was found to have high blood pressure who had not been previously diagnosed. Only 4 of the 13hypertensive patients had serious associated vascular disease; 3 had coronary vascular disease and 1 had undergone femoral artery bypass surgery.

Other associated problems, each occurring in only 1 patient, included chronic leukemia, asthma, migraine, rheumatoid arthritis, and amyotropic lateral sclerosis. The patient with migraine experienced her permanent visual loss in association with a headache.

Associated ocular diseases

Three of the patients (cases 11, 16, 33) had intracapsular cataract extraction with the placement of an intraocular lens, and 2 other patients (cases 2, 25) had an intracapsular cataract extraction without pseudophakos. Two of these patients (cases 25, 33) had perioperative visual loss and the other 3 (cases 2, 11, 16) had visual loss several months later. One of the patients (case 2) had been treated for a retinal detachment. Another patient (case 1) had extracapsular cataract extraction with placement of a posterior chamber intraocular lens; visual loss occurred 3 months after surgery. Six patients were on treatment for glaucoma, none of whom had elevated intraocular pressure at the time of consultation. One patient (case 35) developed ischemic optic neuropathy 3 days after laser iridotomy for narrow-angle glaucoma.

Ophthalmologic findings

Visual acuity. Acuities ranged from 20/15 to hand motion. Vision was 20/400 or worse in 29.8% (14 of 47) of the eyes, 20/100 to 20/200 in 19.1% (9 of 47) of the eyes, 20/50 to 20/80 in 29.8% (14 of 47) of the eyes, and 20/40 or better in 21.3% (10 of 47) of the eyes.

Color vision. The American Optical (AO) color plates were used to test color appreciation. In 26 of 47 eyes, (55.4%) the patient was unable to identify more than 5 of the 15 plates; 34% (16 of 47 eyes) were able to identify between 6 and 10 plates, and only 10.6% (5 of 47 eyes) were able to identify more than 10 plates. For comparison, 85.2% (23 of 27) uninvolved fellow eyes were able to identify more than 10 plates.

Afferent pupillary defect. Twenty-eight of the 37 patients were found to have a relative afferent pupillary defect. Lack of pupillary defect was due to bilateral ocular involvement in 5 patients and to minimal field involvement in 2 (cases 17, 20). Information was unavailable in 2 cases.

Intraocular pressures. The Goldmann applanation tonometer was used to obtain measurements of intraocular pressure prior to pupillary dilation. Pressures ranged from 12 to 22 mm Hg (average 17.2 mm Hg), including the 6 patients with treated glaucoma.

Bilaterality. Ten patients had bilateral disease. Eight had developed ischemic optic neuropathy in the first eye prior to being seen in consultation by us; 2 patients developed step-wise visual loss in their second eye while under our observation.

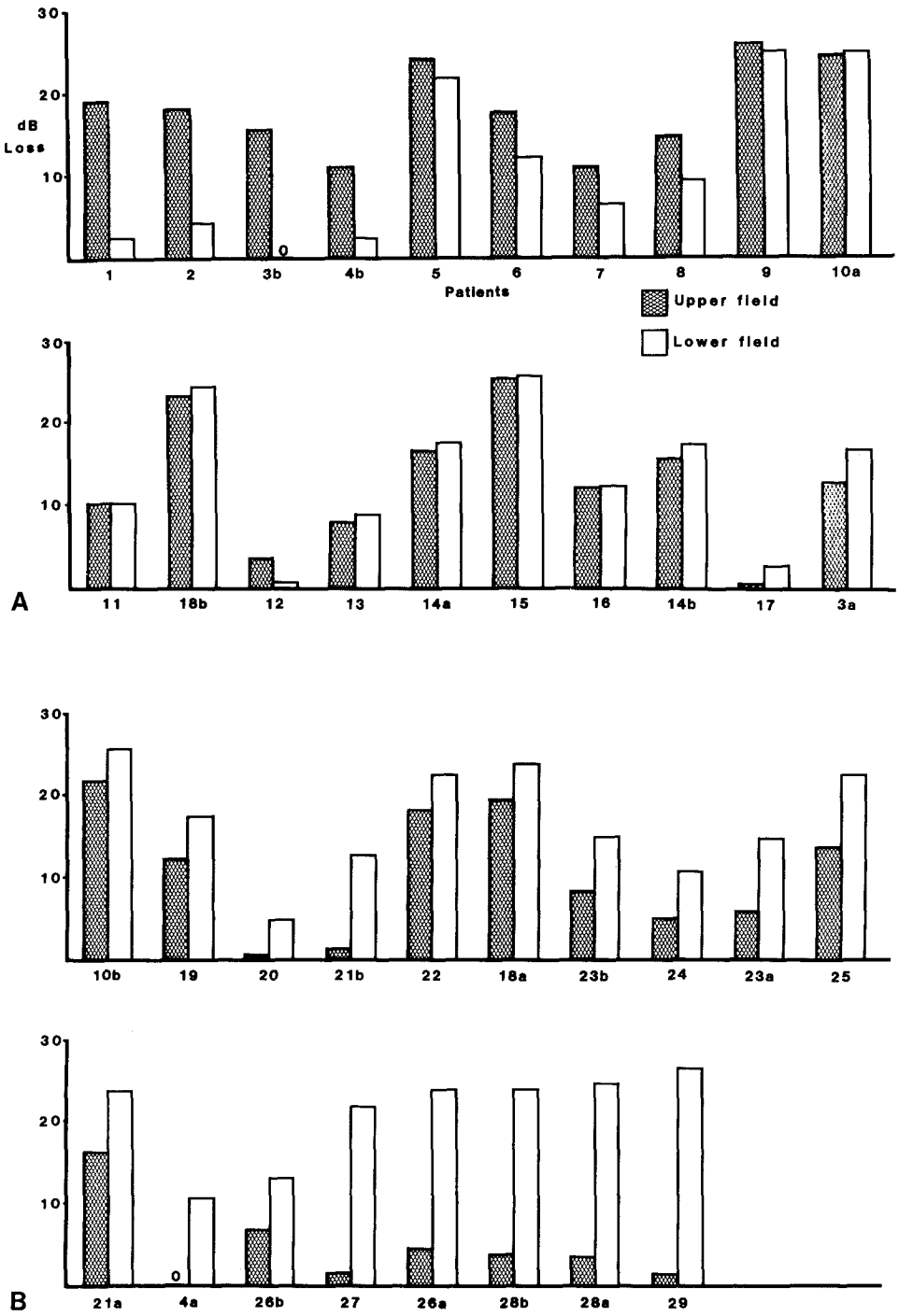

Fig. 2A, B. This series of graphs compares absolute visual fields (dB) of upper (*shaded*) and lower (*white*) field loss for each patient. The visual fields are arranged from greatest upper field loss to greatest lower field loss. Whether or not altitudinal character is present, some field loss was apparent in both upper and lower fields for almost all patients

Analysis of visual fields

Determination of altitudinal characteristics of field loss. In Fig. 1, visual fields are ordered from greatest upper-field loss to greatest lower-field loss. Statistical verification of altitudinal character was established by using *t*-tests to compare upper and lower mean field loss, assuming the sensitivities of each hemifield to be independent. On this basis, 55.3% of the fields were altitudinal at the $P<0.05$ level. Although $t>2.91$ established significance at this level, a larger *t*-value suggested a higher degree of altitudinal character for any particular field (see fields 1–4b and 27–29). Despite the large number of visual fields with significant altitudinal loss, all but 2 (3b, 4a) showed some sensitivity loss in both upper and lower fields (Fig. 2).

When the degree of altitudinal character (*t*-value) was evaluated as a function of loss of visual sensitivity for the entire field (dB loss), the highest *t*-values occurred with loss of sensitivity between 5 and 20 dB. This result was not unexpected since total loss in one hemifield with no loss in the companion hemifield would together average as an intermediate full field loss. Similarly, minimal average full-field loss could not occur if involvement of a single hemifield were extensive, and severe average full-field loss could not occur unless involvement of both hemifields was extensive.

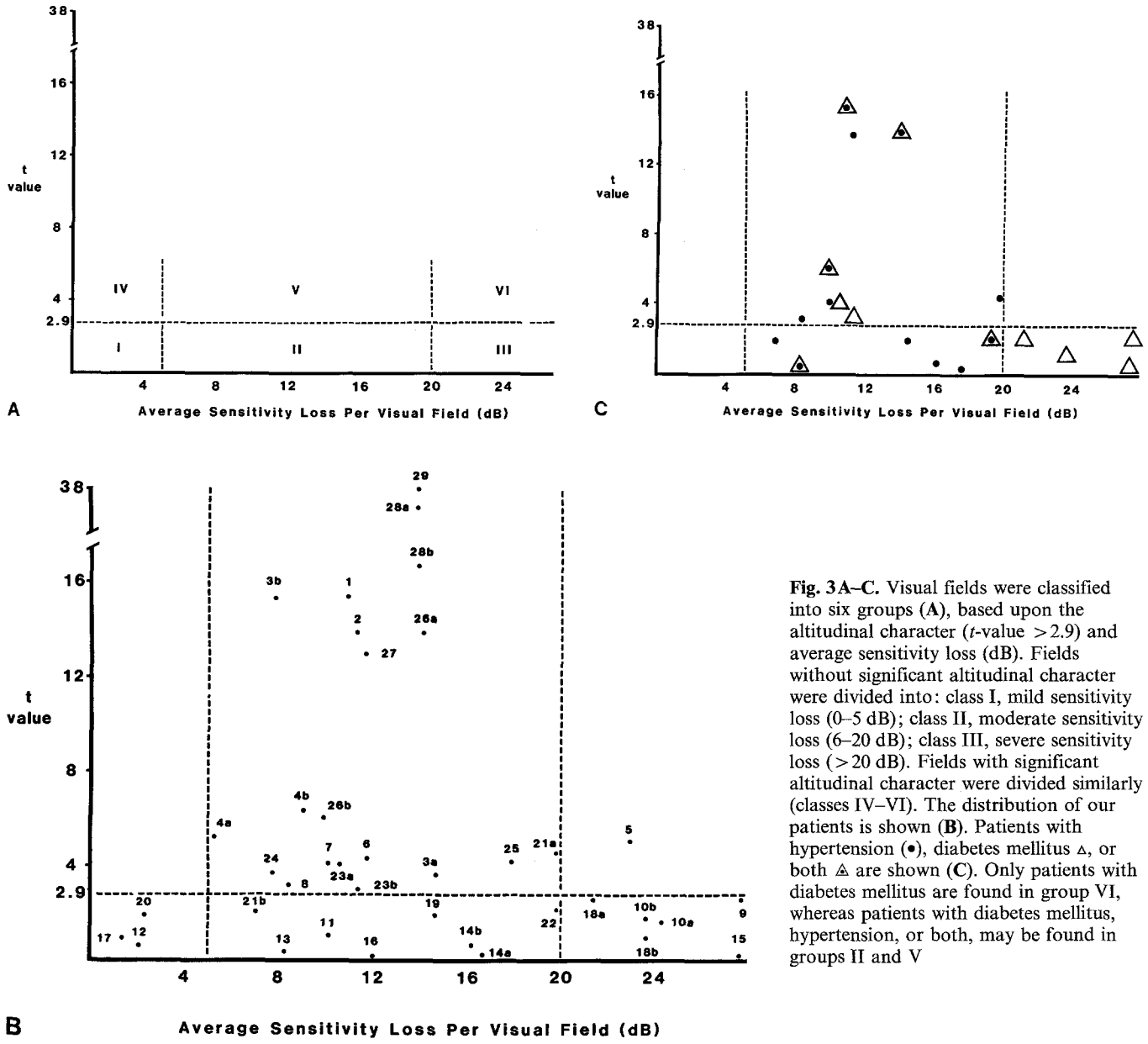

Fig. 3A–C. Visual fields were classified into six groups (**A**), based upon the altitudinal character (t-value >2.9) and average sensitivity loss (dB). Fields without significant altitudinal character were divided into: class I, mild sensitivity loss (0–5 dB); class II, moderate sensitivity loss (6–20 dB); class III, severe sensitivity loss (>20 dB). Fields with significant altitudinal character were divided similarly (classes IV–VI). The distribution of our patients is shown (**B**). Patients with hypertension (•), diabetes mellitus △, or both ◬ are shown (**C**). Only patients with diabetes mellitus are found in group VI, whereas patients with diabetes mellitus, hypertension, or both, may be found in groups II and V

Six classes (Fig. 3a) of visual field loss in anterior ischemic optic neuropathy could be identified by using full-field loss of sensitivity (mild, 0–4 dB; moderate, 5–20 dB; severe, >20 dB) and significant altitudinal loss ($t>2.91$) as criteria. Visual fields classified into groups I–III had no significant altitudinal character and demonstrated mild, intermediate, or severe average sensitivity loss, respectively. Visual fields classified into groups IV–VI had significant altitudinal character and demonstrated mild, intermediate, or severe loss, respectively. Visual fields of most patients were identified in groups II, III, and V (Fig. 3b). Statistical comparisons of clinical features were then possible among these classes using the Fisher Exact Test. Patient age, visual acuity, and color vision did not vary between groups.

Significant clustering ($P<0.02$) of field defects into groups II and V for hypertensive individuals and hypertensive diabetics, and into group III for diabetics, was identified. This clustering is shown graphically in Fig. 3c. Overall, diabetics constituted 75% of patients with visual fields in group III, compared to 10% in group V. Hypertensive diabetic patients constituted 25% of fields in group II and 15% of fields in group V. Hypertensive patients constituted 50% of fields in group II and 20% of fields in group V.

Analysis of concentric regions of visual fields

Those patients without altitudinal field defects of statistical significance were evaluated for possible central scotoma by comparing the central 10° of field with the entire central field, using the t-test. No fields were found to be significantly denser centrally at the $P<0.005$ level, although one field (case 16) was significant at $P<0.05$.

Except for one patient (case 3a) with a single quadrant of arcuate involvement and one patient (case 20) with combined centrocecal and arcuate defect, the remainder of the fields without statistically significant altitudinal defects had diffuse field loss.

Table 2. Comparison of series

Series	Patients	Peak age	Mean age	M	F	F:M	DM	HBP	Bilaterality	Visual field defect
[8]	48	55–65		22	26	1.2:1	38%	26%	48%	80% Arcuate
[3]	37	56–70				1:1		44%	38%	69% Arcuate
[7]	40		57						42%	
[9]	24						10%	11%		
[6]	19	64–85	75	6	13	2.1:1	5%	47%	10%	16% Altitudinal 47% Diffuse
[20]	11	60–79	59.5	7	4	0.5:1	54%	45%	64%	Altitudinal
[10]	212	50–79	63	100	112	1.1:1	16%	41%		
Present series	37	50–69	61.9	16	21	1.3:1	24%	35%	27%	55% Altitudinal 40% Diffuse

Regions between 30° and 60° from fixation were quantitatively assessed in 28 fields; in only one patient (case 23) did the peripheral field change a patient's classification (from altitudinal to diffuse loss). Therefore, the peripheral fields were excluded from further statistical consideration.

Discussion

Clinical features of idiopathic anterior ischemic optic neuropathy

The clinical characteristics of patients in the present study of anterior ischemic optic neuropathy compared favorably with those from other series [3, 6–10, 20]. These clinical data are compared in Table 2. The differences in the patterns of visual field loss reported by various authors may be secondary to problems of definition.

Although all patients were classified as idiopathic because they were not arteritic, some histories suggested coincident ocular or systemic conditions. Cataract extraction, which was our most frequently encountered coexisting ocular problem, was first associated with AION by Townes and colleagues [24] and later by Reese and Carroll [23]. An increase in postoperative intraocular pressure was cited by Hayreh [13] as predisposing to optic nerve injury, but this occurred in only one of our patients. Patients with intracapsular surgery seemed more prone to developing optic neuropathy. Extracapsular surgery is more common than intracapsular surgery in our locale, but only one of the six eyes had undergone an extracapsular procedure. A larger study is needed to determine whether extracapsular surgery is protective against the development of postcataract ischemic optic neuropathy.

Glaucoma was diagnosed in six patients, but the lack of pressure elevation at the time of onset of optic neuropathy probably argues against a precipitating role, as was suggested by Foulds [9].

A procedure not previously reported to be associated with optic neuropathy was laser iridotomy, of which we had one case. Though possibly a coincidence, this complication could result from the transmission of large amounts of laser energy to the vasculature of the optic nerve [15].

The systemic risk factors of diabetes and hypertension are discussed below. The only other previously identified risk factor associated with ischemic optic neuropathy was a single case occurring with migraine [5, 19, 25].

Classification of visual field defects in the understanding and management of ischemic optic neuropathy

Although most studies, including this one, identify hypertension and diabetes as potential systemic risk factors (see Table 2), their specific effects on the clinical presentation of AION have been investigated previously only by Eagling and co-workers [7]. They placed patients with these two diseases into separate but heterogeneous groups. The group that included diabetes demonstrated a 71% incidence of diffuse visual field loss, distinct from the 67% incidence of altitudinal field loss in the group that included hypertension. By utilizing automated (Octopus) static perimetry as a new tool for clinical research, we have been able to associate diabetes specifically with visual field loss, which was severe and without altitudinal character. Patients with both diabetes and hypertension tended to demonstrate altitudinal field loss. The presence of severe diffuse field loss should indicate the possibility of undiagnosed diabetes mellitus.

The association of specific patterns of visual field loss with specific systemic diseases suggests that different pathophysiologic mechanisms may produce optic nerve damage that fulfills the clinical criteria for anterior ischemic optic neuropathy. The severe diffuse visual loss of our diabetic patients argues against localized vascular occlusion as the etiology, as was suggested by Hayreh [11]. More likely, the entire capillary net of the optic nervehead is affected, either by microangiopathy or possibly by vitreous traction [21]. The more focal visual field loss of our hypertensive patients is consistent with the hyaline arteriosclerosis that occurs in this disease. However, even patients with highly altitudinal field defects demonstrated some loss of sensitivity in the less affected hemifield, suggesting some generalized vascular involvement. Therefore, our clinical findings are consistent with the experimental studies of Anderson and Davis [2], which demonstrated the need for multiple vessel involvement to produce ischemic disease of the otpic nervehead.

In summary, the categorization of visual field defects in anterior ischemic optic neuropathy may be correlated with systemic etiologic mechanisms. However, further studies are desirable to confirm and extend these findings. In the future, quantitative analysis of visual fields may be useful in evaluating the conditions under which systemic corticosteroids or other therapeutic manipulations may be efficacious.

Acknowledgement. This study was supported in part by an award from Research to Prevent Blindness, Inc.

References

1. Anderson DR, Braverman S (1977) Re-evaluation of the optic disk vasculature. Am J Ophthalmol 82:165–174
2. Anderson DR, Davis EB (1974) Retina and optic nerve after posterior ciliary artery occlusion. An experimental study in squirrel monkeys. Arch Ophthalmol 92:422–426
3. Boghen DR, Glaser JS (1975) Ischemic optic neuropathy. The clinical profile and natural history. Brain 98:689–708
4. Cogan DG (1966) Neurology of the visual system. Thomas, Springfield, pp 137, 173, 185–188
5. Cowan CL Jr, Knox DL (1982) Migraine optic neuropathy. Ann Ophthalmol 14:164–166
6. Cullen JF (1967) Ischemic optic neuropathy. Trans Ophthalmol Soc UK 87:759–774
7. Eagling EM, Sanders MD, Miller SJH (1974) Ischemic papillopathy. Clinical and fluorescein angiographic review of forty cases. Br J Ophthalmol 58:990–1008
8. Ellenberger C Jr, Keltner JL, Burde RM (1979) Acute optic neuropathy in older patients. Arch Neurol 28:182–185
9. Foulds WS (1969) Visual disturbances in systemic disorders optic neuropathy and systemic disease. Trans Ophthalmol Soc UK 89:125–146
10. Guyer DR, Miller NR, Aver CL, Fine SL (1985) The risk of cerebrovascular and cardiovascular disease in patients with anterior ischemic optic neuropathy. Arch Ophthalmol 103:1136–1142
11. Hayreh SS (1970) Pathogenesis of visual field defects. Role of the ciliary circulation. Br J Ophthalmol 54:289–311
12. Hayreh SS (1975) Anterior ischemic optic neuropathy. Springer, Berlin Heidelberg New York, p 14
13. Hayreh SS (1980) Anterior ischemic optic neuropathy. IV. Occurrence after cataract extraction. Arch Ophthalmol 98:1410–1416
14. Henkind P, Charles NC, Pearson J (1980) Histopathology of ischemic optic neuropathy. Am J Ophthalmol 69:78–90
15. Kaluzny J (1980) Wave movement during argon laser iris coagulation: methods and initial results. Ann Ophthalmol 12:160–164
16. Knox DL, Duke JR (1971) Slowly progressive ischemic optic neuropathy: a clinicopathologic case report. Trans Am Acad Ophthalmol Otolaryngol 75:1065–1068
17. Liberman MF, Shahi A, Green WR (1978) Embolic ischemic optic neuropathy. Am J Ophthalmol 86:206–210
18. Lieberman MF, Maumenee AE, Green WR (1976) Histologic studies of the vasculature of the anterior optic nerve. Am J Ophthalmol 82:405–423
19. McDonald WI, Sanders MD (1971) Migraine complicated by ischemic papillopathy. Lancet II:521–523
20. Miller GL, Smith JL (1966) Ischemic optic neuropathy. Am J Ophthalmol 62:103–115
21. Miller NR (1982) Walsh and Hoyt's clinical neuroophthalmology, 4th ed, vol 1. Williams & Wilkins, Baltimore, p 223
22. Octopus visual field atlas (1978) Interzeag AG, Schlieren, Switzerland
23. Reese AB, Carroll FD (1958) Optic neuritis following cataract extraction. Trans Am Acad Ophthalmol Otolaryngol 62:765–770
24. Townes CD, Moran CT, Pfingst HA (1951) Complications of cataract surgery. Trans Am Acad Ophthalmol Otolaryngol 49:91–107
25. Weinstein JM, Feman SS (1982) Ischemic optic neuropathy in migraine. Arch Ophthalmol 100:1097–1100
26. Zuehlke G, Schmied U (1979) Introduction into technique and clinical application of Octopus perimetry. Proceedings, First International Meeting on Automated Perimetry System – OCTOPUS. Interzeag AG, Schlieren, Switzerland, pp 4–23

Received April 22, 1987 / Accepted October 31, 1987